Behavioural Brain Research 333 (2017) 218–224

Contents lists available at ScienceDirect

Behavioural Brain Research

journal homepage: www.elsevier.com/locate/bbr

Original Article

The ADRA2B gene in the production of false memories for affective information in healthy female volunteers

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ARTICLE INFO

Keywords:
Noradrenaline
Memory distortion
Emotion
Genotype

ABSTRACT

False memories are common memory distortions in everyday life and seem to increase with affectively connoted complex information. In line with recent studies showing a significant interaction between the noradrenergic system and emotional memory, we investigated whether healthy volunteer carriers of the deletion variant of the ADRA2B gene that codes for the α2b-adrenergic receptor are more prone to false memories than non-carriers. In this study, we collected genotype data from 212 healthy female volunteers; 91 ADRA2B carriers and 121 non-carriers. To assess gene effects on false memories for affective information, factorial mixed model analysis of variances (ANOVAs) were conducted with genotype as the between-subjects factor and type of memory error as the within-subjects factor. We found that although carriers and non-carriers made comparable numbers of false memory errors, they showed differences in the direction of valence biases, especially for inferential causal errors. Specifically, carriers produced fewer causal false memory errors for scripts with a negative outcome, whereas non-carriers showed a more general emotional effect and made fewer causal errors with both positive and negative outcomes. These findings suggest that putatively higher levels of noradrenaline in deletion carriers may enhance short-term consolidation of negative information and lead to fewer memory distortions when facing negative events.

1. Introduction

When memories deviate from what truly happened, diverse types of errors, including false memories, may arise (e.g. [1–4,14]). False memories are common in everyday life but the most remarkable aspect of these memory errors is that individuals not only claim that these memories are familiar, but they affirm to recollect contextual and temporal details associated with the encoding of the information. Most importantly, research on false memories has led to important contributions towards understanding normal memory functions (e.g. [5–9]), memory failures in specific brain diseases (e.g. [10]), and has highlighted the importance of considering diverse features that accompany an event in memory.

The main explanation put forward to account for the generation of false memories refers to the semantic elaboration hypothesis which suggests that false memories increase as the semantic elaboration of to-be-remembered information increases [11,12]. For example, developmental studies [13] found that children, who typically have reduced semantic processing and do not spontaneously form interconnected meanings unless the information being encoded is consistent with the gist of experience, were less prone to false memories. Ref. [13] summarized global age trends for data from published experiments using the DRM task. In this task, individuals who study a list of words followed by either a free recall or recognition task, generally show higher levels of false recognition for distracters that represent semantic associations of the studied lists [14]. Ref. [13] found that false memories generally increased from the age of 5 on in recall tasks and after the age of 7 for recognition tasks. Moreover, studies on text comprehension that adopt more complex stimuli (i.e., stories) emphasize that when reading stories, semantic elaboration also drives people seek to identify causal factors and to make inferences in order to link characters and events (e.g., [15] and to achieve text coherence. This search for coherence may subsequently enhance memory errors and lead to an increase in false memories. Differently, false memories may decrease if supported by abilities such as source monitoring, reductions in reliance on gist traces, and/or through effective metacognitive strategies. Indeed, studies have shown how younger and older adults are more prone to false memories as item-specific encoding decreases and gist encoding increases [16].

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http://dx.doi.org/10.1016/j.bbr.2017.06.031
Received 12 January 2017; Received in revised form 15 June 2017; Accepted 19 June 2017
Available online 08 July 2017
0166-4328/

False memories also seem to interact with the affective content of presented material [17]. False memories increase for mood congruent stimuli [18] but are reduced following sleep [19]. Furthermore, a recent series of research on false memories for affective content (e.g. [20–22] with complex stimuli such as scripts rather than single words, showed that false memories not only increased, but were also enhanced by the concurrent activation of self-relevant affective memories.

In particular, crucial to understanding how emotion may affect the generation of false memories is the phenomenon that emotional stimuli are better remembered than neutral ones (the so-called emotional enhancement effect). A series of studies have shown that both valence and arousal of stimuli contributed to the emotional enhancement effect. For instance, Kensinger and Corkin (2004) proposed that the emotional enhancement effect dependent on arousal was associated with automatic encoding processes, whereas the emotional enhancement effect dependent on valence was associated with controlled encoding processes. This may explain why the effect is found in some studies but is absent in others. Others, such as Carstensen et al. (e.g., [23]) explained this emotional advantage in terms of a selectivity towards the pursuit of emotional goals linked to the awareness of the proximity of the life span. This selectivity, in turn, generates a cognitive shift towards emotion processing that boosts memory processes for affective information in general and especially for arousing self-relevant information [24]. Finally, and most relevant to our study, research in neuroscience suggests that the emotional enhancement effect may also be linked to the presence of the genetic variants [25]. In particular, neurobiological models sustain that emotion influences memory via amygdala modulation of the hippocampus and other regions of episodic memory networks and that the locus coeruleus (LC)-norepinephrine (NE) system is crucial for emotion-cognition interactions [26]. The locus coeruleus (LC) is located in the posterior area of the brainstem and receives inputs from the amygdala as well as ventral prefrontal regions and facilitates response to behavioral and biological relevant information while suppressing response to irrelevant information. Importantly, affectively arousing stimuli induce LC phasic activity and enhance encoding, irrespective of whether stimuli are positive [27,28] or negative [29]. This, in turn, further interacts with the influence of NE on longer-term memory processes and leads to enhanced memory for arousing information [30]. Indeed, whether an event is remembered or not depends on modulations in the strength of communication across synapsis and the modulation of NE release following arousing information [31].

In this regard, genetic variations linked to NE has been shown to influence LC activity and convergent evidence suggests that a gene variant, the so-called ADRA2b, is associated with higher levels of intercellular NE availability [32]. The intronless gene ADRA2B, located on the 2p13-q13 chromosome, encodes a seven-pass transmembrane protein widely distributed in the human central and peripheral nervous systems. ADRA2B protein is a subtype of alpha 2-adrenergic receptor (a2-AR) mediating biological effects of endogenous catecholamines, epinephrine and norepinephrine [33]. The presence of the ADRA2B functional polymorphism, consisting of an inframe deletion of three glutamic acids residues (301–303) in the third intracellular loop, leads to a small decrease in coupling receptor efficiency (e.g. [34]). In particular, studies show that administration of NE into the BLA both prior to and following encoding of an event in rats is associated with enhanced memory (for review see Ref. [35]). In humans, the influence of arousal on both encoding and post-encoding processes has been demonstrated by injecting epinephrine or exposing participants to emotionally arousing images prior or after encoding [36].

Again, studies on the interaction between this ADRA2B variant and emotion, with a specific focus on valence effects, show a complex picture (for a review see Ref. [37]). Some studies have found a bias for negative information (e.g. [38,39]) and an association with suicidal behavior [40] whereas others have detected a more general emotion effect since carriers were sensitive to both positive and negative stimuli (e.g. [41–43,25,37]), but all seem to indicate a relevant role of ADRA2B in affective information processing and highlight the importance of investigating genotype-differences using different behavioral paradigms and populations.

Consequently, the present study aims to investigate how affective content influences the generation of false memories as well as the role of ADRA2B in this emotion-memory interaction in healthy female volunteers. In line with recent studies showing the role of noradrenergic neurotransmission in emotion processing, especially in terms of a functional deletion variant of ADRA2B (e.g. [39,25]), we expect affective false memories to be susceptible to ADRA2B genotype differences. This may help clarify the neurobiological mechanisms underlying the generation of affective false memories.

Here, we adopted a paradigm originally proposed by Ref. [44], modified to include affective (both negative and positive) as well as neutral information, to investigate affective false memories and the effect of genotype differences in a group of female volunteers. In this paradigm, we used pictorial representations of daily routines (scripts) to investigate two diverse types of memory errors that can occur while searching for script coherence and comprehension: plausible script errors (participants say “yes” to an unseen picture representing a plausible action in the script) and inferential causal errors (participants say “yes” to an unseen picture depicting the cause of the outcome of the episode). Plausible script errors are an index that participants did process the scripts while inferential causal errors allow us to examine memory distortions determined by inferences made during script elaboration. For example, the bike script depicts the story of a girl on a bike. If participants correctly understand and encode the scripts, they may falsely recognize never seen pictures whose content is coherent with a possible cause of the outcome of the script and show a corresponding increase in the number of causal script errors. That is, they may falsely recognize a trigger picture (i.e. the girl about to cross the road) as one that had been presented and consequently produce a causal error.

In line with semantic elaboration theories and neurobiological models, we expect both deletion carriers and non-carriers to make more inferential errors than plausible script errors since inferential errors derive from erroneous inferences that depend on semantic elaboration processes. Moreover, inferences made while searching for script coherence are based on past personal experience and may ultimately increase arousal levels. In addition, if ADRA2B deletion carriers have higher levels of extracellular NE, we may find different patterns of performance according to affective outcome and arousal. If ADRA2B genotype effects are characterized by a robust negativity bias [39], we expect ADRA2B carriers to make a higher number of errors when scripts contain negative outcomes compared to positive and neutral outcomes. Differently, we might find that carriers make fewer false memories with negative outcomes. Such results would indicate that ADRA2B carriers are able to regulate their memory encoding processes and can modulate their preference for negative information as found in the study by Ref. [45]. In this study, although ADRA2B carriers preferred the story read with a negative prosody, recognition was better for positive stimuli. Alternatively, if ADRA2B genotype effects are characterized by a more general emotion effect, we expect ADRA2B carriers to generate fewer false memories when the scripts contain negative and positive outcomes with respect to neutral ones. Altogether, these findings will thus help disentangling the role of emotion in the generation of false memories (i.e. whether emotion increases or decreases the number of memory errors) and indicate a specific effect of genotype differences in emotional false memories.

2. Method

2.1. Ethics statement

The study was approved by the Departmental Ethics Committee at